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<u>L2</u>	splice adj site	6075	<u>L2</u>
<u>L1</u>	splice adj donor and splice adj acceptor	1308	<u>L1</u>

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- ☐ 1. [20040009603](#). 27 Jan 03. 15 Jan 04. Antiviral vectors. Kingsman, Alan John, et al. 435/456; 424/93.2 435/235.1 435/320.1 A61K048/00 C12N015/867 C12N007/00.
-
- ☐ 2. [20030147907](#). 29 May 01. 07 Aug 03. Retroviral vectors. Kingsman, Alan John, et al. 424/187.1; 424/93.2 435/320.1 435/456 A61K048/00 A61K039/21 C12N015/867.
-
- ☐ 3. [20030104620](#). 25 Nov 97. 05 Jun 03. NON-NATIVE POLYMERASE ENCODING NUCLEIC ACID CONSTRUCT. RABBBANI, ELAZAR, et al. 435/455; 435/320.1 435/325 435/440 435/442 435/91.1 436/6 514/44 536/23.1 536/24.3 536/24.5 C12N015/09 C07H021/04 C12N015/85 C12Q001/68 C12N015/00.
-
- ☐ 4. [20030104611](#). 01 Jun 01. 05 Jun 03. Feline immunodeficiency virus gene therapy vectors. Johnston, Julie C., et al. 435/320.1; 424/199.1 435/235.1 435/325 A61K039/12 C12N007/00 C12N007/01 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74 C12N005/00 C12N005/02.
-
- ☐ 5. [20030087434](#). 25 Nov 97. 08 May 03. PROCESS FOR SELECTIVE EXPRESSION OF NUCLEIC ACID PRODUCTS. RABBANI, ELAZAR, et al. 435/440; 435/320.1 435/325 435/375 435/442 435/455 435/6 435/91.1 514/44 536/23.1 536/24.1 536/24.5 C07H021/04 C12N005/00 C12N015/01 A61K048/00 A61K031/70.
-
- ☐ 6. [20020068354](#). 01 Mar 01. 06 Jun 02. Feline immunodeficiency virus gene therapy vectors. Johnston, Julie C., et al. 435/235.1; 435/320.1 435/456 530/826 536/23.72 536/24.1 C12N007/01 C12N015/867 C07H021/04 C07K001/00 C12N015/74 C12N015/63.
-
- ☐ 7. [20020048805](#). 15 Jan 99. 25 Apr 02. FELINE IMMUNODEFICIENCY VIRUS GENE THERAPY VECTORS. JOHNSTON, JULIE C., et al. 435/235.1; C12P013/14 C12N007/00 C12N007/01.
-
- ☐ 8. [20010007767](#). 25 Nov 97. 12 Jul 01. NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES. RABBANI, ELAZAR, et al. 435/320.1; 435/243 435/325 435/6 435/91.1 435/91.4 514/44 536/23.1 536/24.1 536/24.5 C07H021/04 C12Q001/68 A61K048/00 C12N015/00 C12N005/02 C12N005/00 C12N001/00 C07H021/02.
-
- ☐ 9. [20010006816](#). 25 Nov 97. 05 Jul 01. NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES. RABBANI, ELAZAR, et al. 435/440; 435/320.1 435/325 435/442 435/455 435/91.1 514/44 536/23.1 536/24.3 536/24.5 C07H021/04 C12Q001/68 C12N015/00 A61K031/70 A01N043/04 C12N015/85 C07H021/02.
-
- ☐ 10. [20010006815](#). 25 Nov 97. 05 Jul 01. NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES. RABBANI, ELAZAR, et al. 435/440; 435/320.1 435/325 435/442 435/455 435/91.1 514/44 536/23.1 536/24.3 536/24.5 C12N015/09 C12Q001/68 C07H021/04 C12N015/85 C12N015/00.
-

- ☐ 11. 20010006814. 25 Nov 97. 05 Jul 01. NOVEL PROPERTY EFFECTING AND/ OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES. RABBANI, ELAZAR, et al. 435/440; 435/243 435/252.3 435/320.1 435/325 435/442 435/455 435/91.1 536/23.1 536/24.3 536/24.5 C07H021/04 C12Q001/68 C12N015/01 C12N015/00 C12N015/70 C12N005/02 C12N005/00 C12N015/09 A61K048/00 C07H021/02.
-
- ☐ 12. 6541248. 20 Apr 00; 01 Apr 03. Anti-viral vectors. Kingsman; Alan John, et al. 435/325; 435/320.1 435/455 435/69.1 435/91.4. C12N015/00 C12N015/63.
-
- ☐ 13. 6531123. 25 May 99; 11 Mar 03. Lentiviral vectors. Chang; Lung-Ji. 424/93.2; 424/93.1 424/93.6 435/235.1 435/320.1 435/325 435/366 435/455 435/456 435/457 435/5 435/6 536/23.1 536/23.72 536/24.1. A61K048/00 C12N015/867 C12N015/63 C12N005/10.
-
- ☐ 14. 6312683. 27 Jan 99; 06 Nov 01. Equine infectious anemia virus vectors. Kingsman; Alan John, et al. 424/93.2; 424/93.1 424/93.21 424/93.6 435/320.1 435/325 435/455 435/69.1 514/44 536/23.1. A61K048/00 C12N015/00 C12N015/88.
-
- ☐ 15. 6294165. 18 Dec 97; 25 Sep 01. Defective packaging non-oncoviral vectors based on HIV. Lever; Andrew Michael Lindsay, et al. 424/93.2; 435/320.1 435/325 435/455 435/69.1 514/44. A61K048/00 C12N015/88.
-
- ☐ 16. 6207455. 22 Sep 97; 27 Mar 01. Lentiviral vectors. Chang; Lung-Ji. 435/457; 435/320.1 435/325 435/363 435/366 435/368 435/369 435/370 435/371 435/372 435/455 435/456. C12N015/63 C12N015/867 C12N005/10.
-
- ☐ 17. 6114167. 21 Sep 94; 05 Sep 00. Ribozymes targeting the MoMLV PSI packaging sequence and the HIV tat sequence. Symonds; Geoffrey P., et al. 435/372.3; 435/325 435/366. C12N005/10.
-
- ☐ 18. 5712384. 05 Jan 94; 27 Jan 98. Ribozymes targeting retroviral packaging sequence expression constructs and recombinant retroviruses containing such constructs. Symonds; Geoffrey P., et al. 536/24.5; 435/320.1 435/6 435/91.31 536/23.1 536/23.2. C12Q001/68 C07H021/04 A61K048/00.
-
- ☐ 19. 5693508. 08 Nov 94; 02 Dec 97. Retroviral expression vectors containing MoMLV/CMV-IE/HIV-TAR chimeric long terminal repeats. Chang; Lung-Ji. 435/6; 435/320.1 435/456 435/465 435/69.1 536/24.1. C12N015/00 C12N015/09 C12N015/63 C12P021/06.
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-
- ☐ 1. [20040077577](#). 19 Aug 03. 22 Apr 04. Molecular clones with mutated HIV gag/pol, SIV gag and SIV env genes. Pavlakis, George N.. 514/44; 435/252.33 435/320.1 435/372 530/350 536/23.72 A61K048/00 C07K014/16 C07H021/02 C12N015/867 C12N005/08.
-
- ☐ 2. [20040040052](#). 24 Apr 03. 26 Feb 04. Transgenic organism. Radcliffe, Philippa, et al. 800/21; 435/456 A01K067/00 C12N015/867.
-
- ☐ 3. [20040033595](#). 14 Mar 03. 19 Feb 04. Conditionally replicating vectors for inhibiting viral infections. Humeau, Laurent, et al. 435/320.1; 424/207.1 536/23.72 C12N015/63 C12N015/09 C12N015/00 C12N015/74 C12N015/70 A61K039/21 C07H021/04.
-
- ☐ 4. [20040009603](#). 27 Jan 03. 15 Jan 04. Antiviral vectors. Kingsman, Alan John, et al. 435/456; 424/93.2 435/235.1 435/320.1 A61K048/00 C12N015/867 C12N007/00.
-
- ☐ 5. [20030232058](#). 17 Mar 03. 18 Dec 03. Recombinant alphavirus-based vectors with reduced inhibition of cellular macromolecular synthesis. Dubensky, Thomas W. JR., et al. 424/186.1; 435/235.1 435/456 514/44 536/23.72 A61K039/12 C07H021/02 C12N007/00 C12N015/86.
-
- ☐ 6. [20030147907](#). 29 May 01. 07 Aug 03. Retroviral vectors. Kingsman, Alan John, et al. 424/187.1; 424/93.2 435/320.1 435/456 A61K048/00 A61K039/21 C12N015/867.
-
- ☐ 7. [20030104611](#). 01 Jun 01. 05 Jun 03. Feline immunodeficiency virus gene therapy vectors. Johnston, Julie C., et al. 435/320.1; 424/199.1 435/235.1 435/325 A61K039/12 C12N007/00 C12N007/01 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74 C12N005/00 C12N005/02.
-
- ☐ 8. [20030096397](#). 18 Feb 00. 22 May 03. Recombinant alphavirus-based vectors with reduced inhibition of cellular macromolecular synthesis. Schlesinger, Sondra, et al. 435/320.1; 435/69.1 435/91.1 435/91.2 C12P021/06 C12P019/34 A61K031/665 A01N057/00 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74.
-
- ☐ 9. [20030087419](#). 30 Oct 01. 08 May 03. Regulated nucleic acid expression system. Chang, Yung-Nien, et al. 435/235.1; 435/173.9 435/239 435/320.1 435/325 C12N013/00 C12N007/00 C12N007/01 C12N007/02 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74 C12N005/00 C12N005/02.
-
- ☐ 10. [20030049229](#). 02 Oct 02. 13 Mar 03. Molecular clones with mutated HIV gag/pol, SIV gag and SIV env genes. Pavlakis, George N.. 424/93.2; 435/235.1 435/252.33 435/366 435/456 536/23.1 A61K048/00 C07H021/02 C12N007/00 C12N015/867 C12N005/08.
-
- ☐ 11. [20030026791](#). 27 Mar 01. 06 Feb 03. Conditionally replicating vectors for inhibiting viral infections. Humeau, Laurent, et al. 424/93.21; 424/93.3 424/93.6 435/235.1 435/320.1 536/23.72 536/24.1 A61K048/00 C12N007/01 C12N015/86 C07H021/04 A01N063/00 C12N015/09 C12N015/70.
-
- ☐ 12. [20030008282](#). 06 Jun 01. 09 Jan 03. Compositions and methods for determining anti-viral drug
-

susceptibility and resistance and anti-viral drug screening. Capon, Daniel J., et al. 435/6; 435/320.1 435/5 C12Q001/68 C12Q001/70 C12N015/869.

☐ 13. 20020147169. 29 Nov 01. 10 Oct 02. In vivo selection method for determining inhibitory RNA molecules. Mitrophanous, Kyri, et al. 514/44; 435/320.1 435/455 A61K048/00 C12N015/00 A61K031/70 A01N043/04 C12N015/09 C12N015/63 C12N015/70 C12N015/74 C12N015/85 C12N015/87.

☐ 14. 20020068354. 01 Mar 01. 06 Jun 02. Feline immunodeficiency virus gene therapy vectors. Johnston, Julie C., et al. 435/235.1; 435/320.1 435/456 530/826 536/23.72 536/24.1 C12N007/01 C12N015/867 C07H021/04 C07K001/00 C12N015/74 C12N015/63.

☐ 15. 20020048805. 15 Jan 99. 25 Apr 02. FELINE IMMUNODEFICIENCY VIRUS GENE THERAPY VECTORS. JOHNSTON, JULIE C., et al. 435/235.1; C12P013/14 C12N007/00 C12N007/01.

☐ 16. 20010036655. 01 Jun 01. 01 Nov 01. Molecular clones with mutated HIV gag/pol, SIV gag and SIV env genes. Pavlakis, George N.. 435/235.1; 424/93.21 435/366 A61K048/00 C12N007/01 C12N005/08.

☐ 17. 20010031257. 15 Sep 95. 18 Oct 01. RECOMBINANT FOAMY VIRUS VECTORS FOR MEDICINAL AND DIAGNOSTIC USES, AND PROCESSES FOR PREPARING RECOMBINANT FOAMY VIRUS VECTORS. MEULEN, VOLKER T., et al. 424/93.21; 435/320.1 514/44 536/23.1 A61K048/00 C07H021/04 C12N015/86.

☐ 18. 6656706. 01 Jun 01; 02 Dec 03. Molecular clones with mutated HIV gag/pol, SIV gag and SIV env genes. Pavlakis; George N.. 435/69.1; 424/93.2 435/252.3 435/320.1 435/325 435/455 435/91.4 514/44 536/23.1. C12P021/06 C12N015/63.

☐ 19. 6592874. 18 Feb 00; 15 Jul 03. Recombinant alphavirus-based vectors with reduced inhibition of cellular macromolecular synthesis. Schlesinger; Sondra, et al. 424/218.1; 424/219.1 435/235.1 435/236. A61K039/193.

☐ 20. 6541248. 20 Apr 00; 01 Apr 03. Anti-viral vectors. Kingsman; Alan John, et al. 435/325; 435/320.1 435/455 435/69.1 435/91.4. C12N015/00 C12N015/63.

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FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 18:21:18 ON 06 JUL 2004

L1 21148 S SPLICE(W)DONOR AND SPLICE(W)ACCEPTOR OR SPLICE(W)SITE
L2 3070 S HIV(3A)REV
L3 87 S L1 AND L2
L4 652 S HIV(4A)PACKAG?
L5 2 S L3 AND L4
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)

=> d au ti so pi ab 1-2 16

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AU Mautino, M. R.; Keiser, N.; Morgan, R. A.
TI Improved titers of HIV-based lentiviral vectors using the SRV-1
constitutive transport element
SO Gene Therapy (2000), 7(16), 1421-1424
CODEN: GETHEC; ISSN: 0969-7128
AB The development of lentiviral vectors that use Rev-independent mechanisms
of nuclear export for their genomic RNA could facilitate the construction
of novel anti-HIV vectors. The authors have improved the titers of
Rev-independent lentiviral vectors having the SRV-1 CTE by mutating the
major splice donor and acceptor sites present in the vector and by
re-localization of the CTE sequences adjacent to the HIV-1 3'LTR. These
two modifications have additive beneficial effects on vector titers and
packaging efficiency. Packaging these CTE+ vectors expressing marker
genes with a **Rev**-dependent **HIV-1** helper vector yields
higher titers than are obtained using a Rev-dependent lentiviral vector.

L6 ANSWER 2 OF 2 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU HelgaMaria C; Hammarskjold M L; Rekosh D (Reprint)
TI An intact TAR element and cytoplasmic localization are necessary for
efficient packaging of human immunodeficiency virus type 1 genomic RNA
SO JOURNAL OF VIROLOGY, (MAY 1999) Vol. 73, No. 5, pp. 4127-4135.
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
WASHINGTON, DC 20005-4171.
ISSN: 0022-538X.
AB Although most reports defining the human immunodeficiency virus type 1
(**HIV-1**) genomic RNA **packaging** signal have focused on
the region downstream of the major 5' **splice site**,
others have suggested that sequences upstream of the **splice
site** may also play an important role, In this study we have
directly examined the role played by the HIV-1 TAR region in RNA
packaging. For these experiments we used a proviral expression system that
is largely independent of Tat for transcriptional activation. This allowed
us to create constructs that efficiently expressed RNAs carrying mutations
in TAR and to determine the ability of these RNAs to be packaged, Our
results indicate that loss of sequences in TAR significantly reduce the
ability of a viral RNA to be packaged. The requirement for TAR sequences
in RNA packaging was further examined by using a series of missense
mutations positioned throughout the entire TAR structure. TAR mutations
previously shown to influence Tat transactivation, such as G31U in the
upper loop region or UCU to AAG in the bulge (nucleotides [nt] 22 to 24),
failed to have any effect on RNA packaging. Mutations which disrupted the
portion of the TAR stem immediately below the bulge also had little
effect. In contrast, dramatic effects on RNA packaging were observed with
constructs containing mutations in the lower portion of the TAR stem,
Point mutations which altered nt 5 to 9, 10 to 15, 44 to 49, or 50 to 54
all reduced RNA packaging 11- to 25-fold. However, compensatory double
mutations which restored the stem structure were able to restore

packaging. These results indicate that an intact lower stem structure, rather than a specific sequence, is required for RNA packaging. Our results also showed that RNA molecules retained within the nucleus cannot be packaged, unless they are transported to the cytoplasm by either Rev/Rev response element or the Mason-Pfizer monkey virus constitutive transport element.

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FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 18:21:18 ON 06 JUL 2004

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L2 3070 S HIV(3A)REV
L3 87 S L1 AND L2
L4 652 S HIV(4A)PACKAG?
L5 2 S L3 AND L4
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)
L7 50 DUP REM L3 (37 DUPLICATES REMOVED)
L8 13 S L7 AND VECTOR

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L8 ANSWER 1 OF 13 MEDLINE on STN
AU Hammarskjold M L; Li H; Rekosh D; Prasad S
TI Human immunodeficiency virus env expression becomes Rev-independent if the env region is not defined as an intron.
SO Journal of virology, (1994 Feb) 68 (2) 951-8.
Journal code: 0113724. ISSN: 0022-538X.
AB The human immunodeficiency virus (HIV) Rev protein functions to facilitate export of intron-containing HIV mRNA from the nucleus to the cytoplasm. We have previously shown that **splice site** recognition plays an important role in Rev regulation of HIV env expression. Here we have further analyzed the effects of **splice sites** on HIV env expression and Rev regulation, using a simian virus 40 late replacement **vector** system. env expression from the **vector** became completely Rev-independent when an excisable intron was positioned upstream of the env region, provided that env was not recognized as an intron. Complete Rev regulation was restored either by the insertion of a 5' **splice site** between the intron and the env open reading frame or by deletion of the 3' **splice site** of the upstream intron. These results show that 5' **splice sites** can function as cis-acting repressor sequence (CRS) elements to retain RNA in the nucleus in the absence of Rev. They also indicate that Rev regulation of HIV env expression is critically dependent on whether the env region is defined as an intron. This strengthens the hypothesis that Rev interacts with components of the splicing machinery to release splicing factors and enable export of the mRNA before splicing occurs.

L8 ANSWER 2 OF 13 MEDLINE on STN
AU Lu X B; Heimer J; Rekosh D; Hammarskjold M L
TI U1 small nuclear RNA plays a direct role in the formation of a rev-regulated human immunodeficiency virus env mRNA that remains unspliced.
SO Proceedings of the National Academy of Sciences of the United States of America, (1990 Oct) 87 (19) 7598-602.
Journal code: 7505876. ISSN: 0027-8424.
AB **rev**-regulated expression of HIV-1 envelope proteins from a simian virus 40 late replacement **vector** was found to be dependent on the presence of a 5' **splice site** in the env mRNA in spite of the fact that this mRNA remains unspliced. When the 5' **splice site** upstream of the env open reading frame was deleted or mutated, expression of envelope protein was lost. RNA analysis of cells transfected with 5' **splice-site** mutants showed a dramatic reduction in the steady-state levels of env mRNA whether or not rev was present. Envelope expression could be restored in one of the 5' **splice-site** mutants by cotransfection with a plasmid expressing a suppressor U1 small nuclear RNA containing a

compensatory mutation. These experiments show that U1 small nuclear RNA plays a direct and essential role in the formation of an unspliced RNA that is subject to regulation by rev.

L8 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
IN Humeau, Laurent; Li, Yuxia; Merling, Randall; Dropulic, Boro; Schonely, Kathy L.
TI Improved conditionally replicating lentivirus **vectors** inhibiting wild-type virus replication and their therapeutic uses
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
AB The present invention provides improved conditionally replicating **vectors** that have improved safety against the generation of replication competent **vectors** or virus. The **vector** is dependent upon an external agent to replicate in a target cell, such as a cell infected by the wild-type virus. The agent may be a gene deleted in the **vector** but not from the wild-type virus, making it dependent upon infection for replication. The **vector** carries a gene for an agent that gives the **vector** a selective advantage over the wild-type virus in the target cells, such as a ribozyme specific to the wild type virus. As the wild-type virus is eliminated, the **vector** stops replicating and is therefore at a lower risk of recombining with wild-type virus. Also disclosed are methods of making, propagating and selectively packaging, modifying and using **vectors**. Included are improved helper constructs, host cells, for use with the improved **vectors** as well as pharmaceutical compns. and host cells comprising the **vectors**, the use of **vector** containing host cells to screen drugs, and methods of using the **vectors** to determine gene function. The methods also include the prophylactic and therapeutic treatment of disease, especially viral infection, and HIV infection in particular. The development of a **vector** based on HIV-1 carrying a ribozyme against the U5 element is demonstrated. The **vector** is made resistant to the ribozyme by changes in the sequence of its U5 element.

L8 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
IN Hobom, Gerd; Menke, Anette
TI Influenza viruses with enhanced transcription and replication capacities comprising RNA polymerase similar to that of fowl plague virus and uses for gene therapy and vaccination
SO Eur. Pat. Appl., 137 pp.
CODEN: EPXXDW
AB The present invention provides human influenza viruses comprising an RNA sequence encoding a modified RNA-polymerase (RNAP). It was found that specific modifications of the RNA sequence encoding the RNAP, in particular the RNAP PB1 subunit - so as to code for a polypeptide having a higher similarity with fowl plague virus strain Bratislava (FPV) RNAP - provides viruses capable of recognition of viral RNA (vRNA) promoter sequence variations (the so called promoter-up variants) leading to an increase in transcription and/or replication initiation rates. The vRNA promoter may comprise the modifications G3A and C8U, or G3C and C8G, preferably G3A, U5C and C8U, or G3C, U5C and C8G in the 3'-terminal region (5'-CCUGUUUCUACU-3' or 5'-CCUGUUUUUACU-3'); and the modifications U3A and A8U in the 5'-terminal region (5'-AGAAGAAUCAAGG-3'). The present invention also provides a process for the preparation thereof, pharmaceutical compns. comprising said human influenza viruses and their use for gene transfer into mammalian cells, for ex vivo gene transfer into antigen-presenting cells, such as dendritic cells, for in vivo somatic gene therapy, or in vivo vaccination purposes. The invention also relates to other non-avian influenza viruses, including equine, porcine influenza viruses.

L8 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
IN Chang, Yung-Nien; Lu, Xiaobin; Slepishkin, Vladimir; Conde, Betty; Davis,

Brian; Yu, Qiao; Yang, Yanping; Merling, Randal; Han, Wei; Ni, Yajin; Li, Yuexia; Dropulic, Boro

TI Non-infective viral **vectors** for therapeutic use that can block replication of of wild-type virus

SO PCT Int. Appl., 116 pp.

CODEN: PIXXD2

AB Viral **vectors** for therapeutic use that lack genes essential for replication in a target cell and that can inhibit the replication of a wild-type virus that may arise by recombination are described. The **vectors** carry a gene for a ribozyme or antisense nucleic acid that will act on a sequence found only in the replication-competent virus and block its replication. Also disclosed are methods of making, propagating and selectively packaging, modifying, and using such **vectors**. Included are improved helper constructs, host cells, for use with the improved **vectors** as well as pharmaceutical compns. and host cells comprising the **vectors**, the use of **vector** containing host cells to screen drugs, and methods of using the **vectors** to determine gene function. The methods also include the prophylactic and therapeutic treatment of disease, especially viral infection, and HIV infection in particular. The construction of an HIV-1-based **vector** that included a gene for a hammerhead ribozyme directed against the U5 region of wild-type HIV-1 is described. The U5 region of the **vector** was modified to resist ribozyme cleavage.

L8 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

AU Mautino, M. R.; Keiser, N.; Morgan, R. A.

TI Improved titers of HIV-based lentiviral **vectors** using the SRV-1 constitutive transport element

SO Gene Therapy (2000), 7(16), 1421-1424

CODEN: GETHEC; ISSN: 0969-7128

AB The development of lentiviral **vectors** that use Rev-independent mechanisms of nuclear export for their genomic RNA could facilitate the construction of novel anti-HIV **vectors**. The authors have improved the titers of Rev-independent lentiviral **vectors** having the SRV-1 CTE by mutating the major splice donor and acceptor sites present in the **vector** and by re-localization of the CTE sequences adjacent to the HIV-1 3'LTR. These two modifications have additive beneficial effects on **vector** titers and packaging efficiency. Packaging these CTE+ **vectors** expressing marker genes with a **Rev**-dependent HIV-1 helper **vector** yields higher titers than are obtained using a Rev-dependent lentiviral **vector**.

L8 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

IN Raybak, Susanna M.; Cara, Andrea; Gusella, Gabriele Luca; Newton, Dianne L.

TI Construction of retroviral **vectors** for delivering viral and oncogenic inhibitors

SO PCT Int. Appl., 63 pp.

CODEN: PIXXD2

AB Cell transformation **vectors** for inhibiting HIV and tumor growth are provided. Optionally, the **vectors** encode RNases A superfamily members such as eosinophil-derived neurotoxin (EDN) and onconase. Cells transduced by the **vectors** and methods of transforming cells (in vitro and in vivo) using the **vectors** are also provided. The viral and oncogene inhibitors are typically linked to a promoter such as retroviral HIV LTR promoters, the CMV promoter, the probasin promoter, and tetracycline-responsive promoters. The method is exemplified by construction of a viral **vector** containing a **HIV Rev**-responsive element, an encephalomyocarditis virus internal ribosome entry site, a first viral inhibitor subsequence (for immunodominant proteins such as as Tat, Gag, or Rev), **splice donor** site subsequence, **splice acceptor** site subsequence, the above mentioned promoter, and the EDN coding sequence.

The **vector** may be packaged in a liposome and its contents transduced into CD34+ hematopoietic stem cells, CD4+ cells, and transferrin receptor+ cells. Claimed **vectors** include pBAR, pBAR-ONC, and pBAR-EDN.

L8 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

AU Schneider, Ralf; Campbell, Mel; Nasioulas, Georgios; Felber, Barbara K.; Pavlakis, George N.

TI Inactivation of the human immunodeficiency virus type 1 inhibitory elements allows Rev-independent expression of Gag and Gag/protease and particle formation

SO Journal of Virology (1997), 71(7), 4892-4903

CODEN: JOVIAM; ISSN: 0022-538X

AB The expression of gag, pol, and env of human immunodeficiency virus type 1 (HIV-1) depends on the presence of the viral Rev protein. This dependence is, at least in part, due to the presence of neg. acting sequences (inhibitory or instability elements [INS]) located within unspliced and partially spliced mRNAs. The pos. interaction of Rev with the Rev-responsive element in these mRNAs counteracts the neg. effects of the inhibitory sequences. Here, we demonstrate that in addition to the previously identified INS1 within p17gag, several other INS elements exist within the gag/pol region of HIV-1. These elements act independently of each other and were eliminated by mutagenesis after the introduction of multiple point mutations not affecting the coding region, leading to constitutive high levels of Gag expression. Expression **vectors** containing an intact or nearly intact p55gag region allowed the production of immature viral particles in mammalian cells in the absence of any other HIV proteins. The introduction of addnl. mutations in the protease region allowed efficient production of Gag/protease, which resulted in processing of the Pr55gag precursor and production of mature Gag particles with a lentivirus-like conical-core structure. The elimination of a newly identified INS element within pol and the previously identified CRS located within int was accomplished by the same methodol. Sequence comparisons of the identified inhibitory elements revealed no apparent homologies and demonstrated that these sequences are not **splice sites**. These results demonstrate that the elimination of INS elements leads to efficient expression of HIV-1 mRNAs in the absence of Rev or any posttranscriptional activating mechanisms.

L8 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

AU Michienzi, Alessandro; Prislei, Silvia; Bozzoni, Irene

TI U1 small nuclear RNA chimeric ribozymes with substrate specificity for the Rev pre-mRNA of human immunodeficiency virus

SO Proceedings of the National Academy of Sciences of the United States of America (1996), 93(14), 7219-7224

CODEN: PNASAA6; ISSN: 0027-8424

AB The in vivo effectiveness of ribozymes strongly depends on the correct choice of the **vector** mol. High levels of expression, stability, active conformation, and correct cellular localization are the most important features for a ribozyme **vector**. We have exploited the utilization of the U1 small nuclear RNA (snRNA) as a **vector** for specifically targeting a ribozyme into the nucleus. The Rev pre-mRNA of human immunodeficiency virus type 1 was chosen as target for testing the activity of the U1-ribozyme. The catalytic core of the hammerhead motif, plus the recognition sequences, substituted the stem-loop III of the U1 snRNA. The resulting construct displays efficient cleavage activity in vitro. In addition, in the in vivo system of Xenopus laevis oocytes, the U1-chimeric ribozyme accumulates in large amts. in the nucleus and produces a considerable reduction of Rev pre-mRNA levels. The Rev-specific ribozyme was also inserted in a derivative of the U1 snRNA mutated in the region of pairing with the 5' **splice site**, such as to match it with the suboptimal splice junction of the Rev precursor. This construct shows more efficient reduction of Rev pre-mRNA in vivo than the wild-type U1 **vector**.

L8 ANSWER 10 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
 AU Kotsopoulou E; Kim V N; Kingsman A J; Kingsman S M (Reprint); Mitrophanous
 K A
 TI A Rev-independent human immunodeficiency virus type 1 (HIV-1)-based
vector that exploits a codon-optimized HIV-1 gag-pol gene
 SO JOURNAL OF VIROLOGY, (MAY 2000) Vol. 74, No. 10, pp. 4839-4852.
 Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
 WASHINGTON, DC 20005-4171.
 ISSN: 0022-538X.

AB The human immunodeficiency virus (HIV) genome is AU rich, and this
 imparts a codon bias that is quite different from the one used by human
 genes. The codon usage is particularly marked for the gag, pol, and env
 genes. Interestingly, the expression of these genes is dependent on the
 presence of the Rev-responsive element (RRE) regulatory system, even in
 contexts other than the HIV genome. The Rev dependency
 has been explained in part by the presence of RNA instability sequences
 residing in these coding regions. The requirement for Rev also places a
 limitation on the development of HIV-based **vectors**, because of
 the requirement to provide an accessory factor. We have now synthesized a
 complete codon-optimized HIV-1 gag-pol gene. We show that expression
 levels are high and that expression is Rev independent. This effect is due
 to an increase in the amount of gag-pol mRNA. Provision of the RRE in cis
 did not lower protein or RNA levels or stimulate a Rev response.
 Furthermore we have used this synthetic gag-pol gene to produce HIV
vectors that now lack all of the accessory proteins. These
vectors should now be safer than murine leukemia virus-based
vectors.

L8 ANSWER 11 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
 AU HelgaMaria C; Hammarskjold M L; Rekosh D (Reprint)
 TI An intact TAR element and cytoplasmic localization are necessary for
 efficient packaging of human immunodeficiency virus type 1 genomic RNA
 SO JOURNAL OF VIROLOGY, (MAY 1999) Vol. 73, No. 5, pp. 4127-4135.
 Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
 WASHINGTON, DC 20005-4171.
 ISSN: 0022-538X.

AB Although most reports defining the human immunodeficiency virus type 1
 (HIV-1) genomic RNA packaging signal have focused on the region downstream
 of the major 5' **splice site**, others have suggested
 that sequences upstream of the **splice site** may also
 play an important role. In this study we have directly examined the role
 played by the HIV-1 TAR region in RNA packaging. For these experiments we
 used a proviral expression system that is largely independent of Tat for
 transcriptional activation. This allowed us to create constructs that
 efficiently expressed RNAs carrying mutations in TAR and to determine the
 ability of these RNAs to be packaged. Our results indicate that loss of
 sequences in TAR significantly reduce the ability of a viral RNA to be
 packaged. The requirement for TAR sequences in RNA packaging was further
 examined by using a series of missense mutations positioned throughout the
 entire TAR structure. TAR mutations previously shown to influence Tat
 transactivation, such as G31U in the upper loop region or UCU to AAG in
 the bulge (nucleotides [nt] 22 to 24), failed to have any effect on RNA
 packaging. Mutations which disrupted the portion of the TAR stem
 immediately below the bulge also had little effect. In contrast, dramatic
 effects on RNA packaging were observed with constructs containing
 mutations in the lower portion of the TAR stem. Point mutations which
 altered nt 5 to 9, 10 to 15, 44 to 49, or 50 to 54 all reduced RNA
 packaging 11- to 25-fold. However, compensatory double mutations which
 restored the stem structure were able to restore packaging. These results
 indicate that an intact lower stem structure, rather than a specific
 sequence, is required for RNA packaging. Our results also showed that RNA
 molecules retained within the nucleus cannot be packaged, unless they are
 transported to the cytoplasm by either Rev/Rev response element or the

Mason-Pfizer monkey virus constitutive transport element.

- L8 ANSWER 12 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Huffman K M; Arrigo S J (Reprint)
TI Identification of cis-acting repressor activity within human
immunodeficiency virus type 1 protease sequences
SO VIROLOGY, (4 AUG 1997) Vol. 234, No. 2, pp. 253-260.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900,
SAN DIEGO, CA 92101-4495.
ISSN: 0042-6822.
- AB Human immunodeficiency virus type-1 (**HIV-1**) **Rev**
overcomes negative elements within viral RNAs to allow expression of gag,
pol, and env. The effect of Rev on protein and RNA expression of HIV-1
protease (PR)-containing constructs was investigated utilizing transient
transfection of COS cells. Rev, through the Rev response element (RRE),
resulted in a large increase in proteolytic activity and cytoplasmic RNA
accumulation. Furthermore, Rev increased the level of total RNA produced
by a PR-containing construct. The increase in cytoplasmic RNA accumulation
in the presence of Rev indicated the presence of cis-acting repressor
sequences (CRS) within the RNA produced by this construct. Therefore,
components of the construct were analyzed for CRS activity. PR sequences
in both sense and antisense orientations exhibited CRS activity. RRE
sequences alone conferred a small CRS effect. Additional CRS activity was
present within an unspliced RNA containing only nef and LTR sequences.
These results indicate a novel form of cis-acting repressor activity
within HIV-1 PR; this activity is exerted regardless of the orientation of
PR and appears to function at the level of cytoplasmic or nuclear RNA
stability. (C) 1997 Academic Press.
- L8 ANSWER 13 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Ernst R K; Bray M; Rekosh D; Hammarskjold M L (Reprint)
TI A structured retroviral RNA element that mediates nucleocytoplasmic export
of intron-containing RNA
SO MOLECULAR AND CELLULAR BIOLOGY, (JAN 1997) Vol. 17, No. 1, pp. 135-144.
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW,
WASHINGTON, DC 20005-4171.
ISSN: 0270-7306.
- AB A common feature of gene expression in all retroviruses is that
unspliced, intron-containing RNA is exported to the cytoplasm despite the
fact that cellular RNAs which contain introns are usually restricted to
the nucleus. In complex retroviruses, the export of intron-containing RNA
is mediated by specific viral regulatory proteins (e.g., human
immunodeficiency virus type 1 [**HIV-1**] **Rev**) that bind
to elements in the viral RNA. However, simpler retroviruses do not encode
such regulatory proteins. Here, we show that the genome of the simpler
retrovirus Mason-Pfizer monkey virus (MPMV) contains an element that
serves as an autonomous nuclear export signal for intron-containing RNA.
This element is essential for MPMV replication; however, its function can
be complemented by **HIV-I Rev** and the Rev-responsive
element. The element can also facilitate the export of cellular
intron-containing RNA. These results suggest that the MPMV element mimics
cellular RNA transport signals and mediates RNA export through interaction
with endogenous cellular factors.

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